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ORIGINAL ARTICLES.

ON THE USE OF OPTOCHIN IN DIFFERENT EXTERNAL EYE DISEASES.*

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The specific action of optochin, especially in pneumococcus infection of the eye and particularly in serpiginous ulcer of the cornea, has in the last two years been often discussed by ophthalmologists and been generally acknowledged.. A complete résumé has been given by Morgenroth (*Berl. Klin. Wochenschr.*, 1914, Nos. 47 and 48).

We have used optochin, too, in various external eye affections and almost always with good results.

I should like to divide these cases as follows: 1. Pneumococcus affections of the eye. 2. Diseases of the lacrimal sac, and 3, Eye affections in which the photophobia was primarily influenced by optochin. The histories of the cases follow.

Of typical pneumococcus ulcers of the cornea we have seen only two cases in the last 6 months.

Case I.—D. M., 36 years old, has for four months an ulcer on the left cornea. Has been thus far treated with drops and fomentations. December 23d, R.E. normal. V=1. L.E. very much irritated. Ciliary injection. Central disc-shaped ulcer with progressing edges. Cornea dim, also, in the periphery. Hypopyon 4 mm. high. Pupil small. Iris hyperæmic. Dim red reflex with ophthalmoscope. V=fingers at ½ m. Lacrimal drainage good. Pneumococci in the smear. Treatment: 1 per cent. optochin every hour; atropin 4 times daily, steaming 3 times,

*Klinische Monatsblætter fuer Augenheilkunde, Vol. LIV, p. 446.

bandage. December 24, hypopyon almost gone, ulcer still progressing. Pupil wide. December 27th, no hypopyon. No progress of ulcer. December 29th, pupil smaller. Scopolamin ointment. December 31st, ulcer considerably smaller, partly covered with epithelium. Cornea otherwise clear. 2 per cent. optochin 3 times daily, after cocain, steaming, scopolamin ointment at night. January 9th, discharged. Ulcer healed. Eye opens almost without irritation. A central dense scar reaching somewhat downward, 2 to 8 mm. in diameter. Well covered with epithelium. Fundus, as far as seen, normal. V=fingers at 5 m.

Case II.—Mrs. D. G., aged 62. For 2 weeks ulcer on right eye, growing worse. Had used drops and salve.

January 10.—R.E. conjunctiva much swollen. Chemosis. Eye closed, severe photophobia. The whole eye much injected. In the centre of the cornea a white disc-like ulcer with progressive margin. Pupil small, reacting. Iris hyperæmic, dull. Hypopyon reaching the lower edge of the pupil. Little red reflex from fundus. Drainage apparatus normal. $V=\frac{1}{\infty}$. Numerous pneumococci in the smear. L.E. normal, 1 per cent. optochin every hour, atropin 4 times, steaming, at night scopolamin ointment, bandage. January 12th, hypopyon considerably smaller. Ulcer no larger. Eye without irritation can be opened. January 13th, progressive margin disappeared. No trace of hypopyon. Ulcer much smaller, corneal periphery clear. V=movements of hand. January 15th, ulcer clean. 2 per cent. optochin 3 times daily, atropin-cocain before it, steaming. January 19th, discharged. Eye freely opened. Some injection of globe. Periphery of cornea clear. Dense central scar. Iris still hyperæmic. Pupil more than medium wide, transverse oval. Anterior synechiæ. Fundus, as far as seen through periphery, normal. V=fingers at 5 m.

These two cases of serpiginous ulcer were followed by a case of pneumococcus conjunctivitis proven by microscope.

Case III.—Mrs. M. J., aged 20. Inflamed eyes for 3 days. July 12th, functional examination impossible on account of severe irritation. Phlyctænular conjunctivitis in both eyes. Cornea clear. Argyrol, 2 per cent. yellow ointment. July 23d, typical pneumococcus conjunctivitis with severe affection of the bulbar conjunctiva. Pneumococci in smear. Much photophobia. Cocain twice, then 5 per cent. optochin. At home noviform ointment. Photophobia continues. Drainage apparatus normal.

July 27th, great improvement. Eyes opened freely. 5 per cent. optochin. July 28th, conjunctivæ almost normal. Photophobia gone. No more optochin. 2 per cent. yellow ointment at home. October 17th, no more symptoms.

In cases of affections of the lacrimal sac we use, as Kuemmel* and others, optochin with success, as the following 3 cases show:

Case IV.—Miss B. B., 24 years old. Has had glands operated on. Inflammation of the lacrimal sac for 6 years. Has been operated on elsewhere, but pus formation continues. L. E. is said to have become red a few days ago and badly swollen since yesterday. November 3d, R.E. free from irritation. L.E. great swelling of the lids and neighborhood of the eye, redness and pain. Pus in palpebral fissure. Eye cannot be opened. Conjunctiva bulbi much injected. Cornea clear. Hot applications. November 4th true phlegmone of the lacrimal sac. Temperature high. Aspirin, hot applications. November 5th, whole face swollen, especially on left side. Much pus on bandage. November 6th, incision. Hot applications. November 8th. Much less swelling but eye cannot be opened. Three times daily two drops of cocain and then one drop of 5 per cent. optochin. November 9th, eye freely opened. Still much pus from lacrimal sac on pressure. Daily syringing with boric solution, then 1 per cent. optochin till November 29. Now but little secretion on pressure. Eye open. November 29th, extirpation of left lacrimal sac since pus continues. December 13th, removal of sutures. December 20th, discharged with 2 per cent. yellow ointment.

Case V.—Mrs. A. D., 23 years old. Five years previously had frontal sinus operation. R.E. inflamed for some days. January 2nd, severe conjunctival irritation, lacrimation. Argyrol and xeroform ointment. January 6th, some pus from right lacrimal sac on pressure; 1 per cent. optochin syringed through it. January 7th, considerable improvement. Still a trace of pus. January 8th, no secretion. Syringing through with optochin is unsuccessful, fluid regurgitates. Argyrol at home. January 10th, no secretion. Argyrol and xeroform ointment. Discharged.

Case VI.—Mrs. D. T., 30 years old. L.E. inflamed for two weeks. October 19th, R. E. macula corneæ. Fundus normal. L.E. upper lid swollen, much irritation, cornea downward infiltrated (keratitis herpetiformis). Fundus normal. Atropin, xeroform-dionin ointment. Hot applications. October 22d,

*Muench. med. Wochenschr., 1914, No. 24.

some pus from lacrimal sac on pressure. Zincum-suprarenin solution and then 1 per cent. optochin syringed through. Fluid passed easily, probe, also. Three times daily atropin-cocain; 3 times 5 per cent. optochin. Bandage. October 23d, fluorescin positive. Much pus can still be pressed out of lacrimal sac. October 26th, lacrimal sac free. Eye much less irritated. Cornea outside of herpes clear. Extirpation of sac deferred. The same medication. November 3d, hordeolum L., cornea dim, globe strongly injected. Lacrimal sac free. Hordeolum cauterized. November 10th, lacrimal sac remains free, passage permeable. Cornea almost clear. Fluorescin negative. No more optochin, 2 per cent. yellow ointment. Discharged. Eye almost without irritation. Small triangular macula corneæ downwards. Fundus normal.

In the *Muenchener med. Woehenschrift* Peterka* published two cases of conjunctivitis exzematosa with severe photophobia. He treated these with 5 per cent. optochin with great success which he ascribes to the anæsthetizing action of optochin. By this we were prompted to use optochin in several eye affections which were accompanied by photophobia.

Case VII.—G. R., 5 years old. Trepanation after Elliot on account of glaucoma with congenital buphthalmus on both sides on May 28th upwards and, since tension rose again to 45 and 40, downwards on June 15th. June 18th, sutures removed. Patient cannot open the eyes on account of severe photophobia. The same June 25th. After two instillations of 5 per cent. optochin both eyes were opened freely. June 30th, photophobia returned, but disappears after 5 per cent optochin instillation. Discharged July 8th, eserin in oil.

Case VIII.—Mrs. M. K., 32 years old. Treated formerly with syrgol for acute follicular conjunctivitis. November 26th, again has severe follicular conjunctivitis in both eyes (suspicion of trachoma). Syrgol drops. November 27th, much photophobia, rubbed with copper sulfate ointment. November 28th fewer follicles, severe photophobia. Twice 5 per cent. optochin after cocain. Xeroform ointment at home. November 30th, much improved. No trace of photophobia. 5 per cent optochin, noviform ointment at home. Is treated further from December 1st to 23d on account of the follicular conjunctivitis. Photophobia did not return.

Case IX.—E. K., 5 years old. Both eyes inflamed for a week.

*1914, No. 22.

Had used boric acid applications. July 13th, both eyes closed, severe photophobia. R.E., two small plyctænulæ at the lower corneal margin. L. cornea clear. Two drops of cocain, then two drops of 5 per cent. optochin. July 14th, both eyes freely opened, no photophobia. Same treatment. Yellow ointment at home. July 18th, both corneæ clear. No photophobia. Discharged August 15th.

Case X.—F. G., 2 years old. July 16th, R.E. very much irritated. Conjunctival and ciliary injection. Central corneal ulcer. Scopolamin ointment, since pupil is very small. At home yellow ointment and ½ per cent. atropin. July 22d, severe photophobia. Cocain, then 5 per cent optochin. July 23d, the same. July 24th, atropin twice daily, cocain followed by 5 per cent. optochin 3 times daily. July 26th, both eyes freely opened. August 1st, optochin left off, patient discharged with atropin and yellow ointment. August 11th, ulcer healed. Eyes opened freely.

Case XI.—M. L., 5 years old. Eyes inflamed for a week. July 10th, R. and L. E. severe phyctænular conjunctivitis. Recent corneal infiltrations. Severe photophobia. Atropin, cocain, then 5 per cent. optochin. July 11th, photophobia gone. Once more the same treatment. At home atropin and 2 per cent. yellow ointment. July 14th, both eyes opened freely. Corneal infiltrations disappearing.

Case XII.—H. H., 7 years. Came on June 30th. Inflamed right eye. Severe conjunctival and ciliary irritation, much photophobia, eyes closed. Fresh central and peripheral infiltrations of the cornea, two phylctænules at the external limbus. Pupil small, iris normal. Scopolamin ointment. At home atropine and yellow ointment. July 6th, photophobia rather increased, otherwise the same. Twice 5 per cent. optochin after cocain. July 7th, photophobia has disappeared. Eye opened freely. Once more 5 per cent. optochin. At home yellow ointment. July 9th, eye perfectly free from irritation.

Case XIII.—W. L., 8 years old. L. E. has been inflamed for some days. November 14th, great irritation, lacrimation and photophobia. Conjunctiva greatly injected. Paracentral corneal infiltration. November 15th, both eyes freely opened. Atropin left off. Optochin twice daily, 2 per cent. yellow ointment. November 17th, photophobia has not returned. Optochin left off. November 20th, discharged. Cornea clear. Eye freely opened.

Case XIV.—H. M., 2½ years old. Both eyes have been inflamed for two weeks and secrete pus. The last two days they could not be opened. November 20th, lids swollen, especially left; great photophobia, fibrinopurulent discharge (no membranes), cornea infiltrated down and outwards. Cocain twice daily, then 5 per cent. optochin, atropin L.E. November 21st, after six drops of 5 per cent. optochin both eyes are opened. Optochin continued, yellow ointment. November 29th, no photophobia. Atropin and optochin left off. December 4th, discharged. No irritation. Macula corneæ L.E.

Case XV.—G. A., 1 year old. Has previously been treated for corneal ulcers. November 13th, marginal phlyctænules on both eyes. Severe photophobia. Three times daily 2 drops of cocain followed by 5 per cent. optochin. November 15th, both eyes open freely. Cocain, optochin and 1 per cent yellow ointment. November 20th, no photophobia. Marginal ulcers almost gone. November 23d, discharged.

Case XVI.—W. V., 11 years old. Both eyes inflamed for 5 days. December 26th, upper lids much swollen. Patient cannot lift them even with a great effort. Conjunctivitis phlyctænularis both, keratitis herpetiformis R.E. Left cornea clear, two phlyctænules at inner limbus. Three times daily cocain followed by 5 per cent. optochin. December 27th, no more photophobia, eyes open freely. December 29th, 2 per cent. optochin and yellow ointment. December 31st, both eyes free from irritation. L. cornea clear, R. small macula. Discharged with 2 per cent. yellow ointment. January 7th, perfectly well.

Case XVII.—F. N., 8 years old. Formerly treated for scrophulous ophthalmia. November 21st, severe blepharoconjunctivitis. Much photophobia. Conjunctiva œdematous. Lid margins eczematous. Corneæ clear. Three times daily cocain followed by 5 per cent. optochin, xeroform ointment. Zinc ointment for lid margins. November 25th, unchanged. The same treatment, bandage. November 26th, photophobia still great till December 4th, when the eyes can be opened for a short time. Xeroform ointment changed to yellow ointment. December 5th, eyes freely opened till December 11th, when optochin was left off. December 12th, discharged.

Case XVIII.—H. A., 14 years old. January 9th, has had inflamed eyes previously. L.E. inflamed for 3 days. R.E. central corneal macula. V=0.5. L.E., much irritation, blepharospasm, corneal ulcer near outer limbus, cornea in its neighborhood dim.

Fingers at 2 m. Pupil small, no view of fundus. Atropin twice in L.E., cocain three times followed by 5 per cent. optochin, steaming. January 10th, can open the left eye for a short time. January 11th, eye opens freely. Treatment the same. Yellow ointment. January 13th, eye continues free from photophobia. Atropin and optochin left off. January 19th, discharged, yellow ointment. V=0.5 to 0.6.

Case XIX.—M. H., 3 years old. January 5th, much inflammation in L.E., R.E. freely opened. Marginal phlyctænules, up and in and out. L. E., blepharospasm, great irritation. Central corneal ulcer. Pupil small. Twice atropin-cocain, followed by 5 per cent. optochin. January 6th, both eyes open, L.E., only for a time, especially in bright light. January 9th, no more photophobia. Atropin left off, yellow ointment. January 12, optochin left off. January 14th, discharged.

Case XX.—F. R., 25 years old. December 19th, inflammation of R.E. 6 weeks old. Much photophobia, central herpetiform keratitis. December 20th, 3 times daily atropin and cocain, followed by 5 per cent. optochin, 3 times thermophor. December 22d, photophobia almost gone. Fluorescin positive. December 29th, no more photophobia. Severe herpes corneæ. Abrasion of cornea followed by subconjunctival injection of asterol. December 30th, photophobia returned. Atropin, cocain and optochin. January 4th, eye open, cornea much clearer. Strong reaction to tuberculin Rosenbach 0.05. Tuberculin cure begun. January 11, discharged. Eye free from irritation. Small central macula. V. with +1=0.67 to 1.0.

CONCLUSIONS.

The cases here reported leave no doubt as to the value of optochin in external eye affections. The prompt effect of a 1 per cent. optochin solution in pneumococcus ulcer of the cornea justifies the use of optochin in all such cases. The therapy of such ulcers is greatly simplified by it. Formerly these were treated in the main by three methods: (1) Saemisch's incision; (2) cauterization, and (3) by iontophoresis. By the first and second methods vision is almost always greatly impaired by the following dense scar; all three of these methods presume special deftness and special apparatus. In optochin the practicing physician has a means in his hand, the easy application of which is of great importance for an early and above all a prophylactic therapy of corneal ulcers in general practice. Of course, a punc-

tual instillation every one or two hours of the optochin solution is imperative and farther progressed will as formerly need oculistic and if possible clinical treatment. The earlier corneal ulcers are treated with optochin, the better the prognosis for vision. This is shown in cases I and II. Both of these patients waited much too long before coming for treatment. The result was a dense corneal scar. Concerning the prompt and certain effect of optochin, our cases agree perfectly with those of Leber,* Schur,† Goldschmidt,‡ and others, who could report a large number of corneal ulcers treated successfully with optochin. Optochin was a great gain in corneal ulcers which were associated with an affection of the lacrimal sac. The instillation of a 1 per cent. solution into the conjunctival combined with syringing through the lacrimal drainage apparatus simplifies the treatment considerably. Since as we saw in cases V and VI the purulent secretion of the sac yielded to the syringing through with optochin, ulcer and blennorrhœa of the lacrimal sac can be treated simultaneously. After the extirpation of the lacrimal sac a pressure bandage was necessary for from 5 to 6 days, a simultaneous corneal ulcer could during these days not be properly treated with atropin, steam, etc. Should, however, the lacrimal sac not be removed at the beginning, the therapeutic success with the serpiginous ulcer would be retarded by the secretion from the lacrimal sac. Thus we were always in a dilemma when both cornea and lacrimal sac had to be treated. In this optochin has produced a marked improvement in the therapy. After optochin has removed the danger of corneal perforation, that is when the ulcer is covered with epithelium, the operative removal of the lacrimal sac can be undertaken and the eye can be bandaged for several days without fear. Thus the syringing through with optochin cannot take the place of the extirpation of the lacrimal sac, because in this way a real cure of the disease of the lacrimal sac cannot be obtained. Sooner or later in consequence of the ectasia of the sac a new infection takes place which makes the extirpation necessary.

The effect of a 5 per cent. optochin solution on photophobia in different external eye affections, especially scrophulous ophthalmia, is really astonishing. One is surprised to see how children who have been unable to open their eyes for days and

*Ophth. Gessllsch., Heidelberg, 1913.
†Klin. Mtsbl. f. Augenhlk., 1913, Oct. and Nov.
‡Klin. Mtsbl. f. Augenhlk., 1913, Oct. and Nov.

weeks suddenly open them freely after a few drops of optochin, and how after a short treatment the photophobia disappears lastingly.

Optochin acts slowly or may altogether fail in such cases of phlyctænular eye affections which are accompanied by eczema of the lids, especially when tears in the skin at the outer canthus by their continued irritation render impossible the opening of the eyes. Before these tears are cured by some ointment optochin can do little good. This we saw in the case of a girl of 12 years who had marginal phylyctænules in both eyes with great photophobia which was at first removed by optochin. When, however, a moist eczema was added to it and the photophobia had returned, the action of optochin failed until the eczema was cured by an ointment. Case XVII, too, proves the retarding of the effect of optochin by eczema of the lids. There were, also, two interesting cases which we treated with optochin without success.

The first case was one of bilateral parenchymatous keratitis (Wassermann+) in a young man of 16 who had suffered for several weeks with severe photophobia and lacrimation. Since optochin treatment failed us and the photophobia did not grow any less, we gave up its use.

The second case was one of lime burn of the cornea and conjunctiva which caused severe photophobia. One drop of a 5 per cent. optochin solution caused much lacrimation. The irritative condition was made considerably worse. The next day optochin was tried again after several instillations of cocain. The effect was the same. It not only was therapeutically without effect, it even made conditions worse, so that we were forced to give it up.

This was an interesting observation that the action of optochin fails in, if we may say so—one kind of photophobia while it is prompt and successful in an other one. It acts almost uniformerly favorably in the photophobia of scrophulous ophthalmia; it was, also, successful in case VII (after an Elliot trepanation) and in case VIII (follicular conjunctivitis). It failed in the case of parenchymatous keratitis and the one of lime burn. This led to the assumption that the action of optochin in photophobia can probably not be traced to the anæsthetizing effect of optochin, as Peterka believes. For this, according to my observation, the optochin anæsthesia seems too weak and too short. At any rate, were it true, the action of a 5 per cent. cocain solution should act even stronger against the photophobia, since the anæs-

thesia from cocain is more intense and lasts longer than that of optochin. Yet with cocain alone we have never observed this, on the contrary with cocain alone there was only a small and short improvement in the photophobia, and an undoubtedly stronger and more lasting effect with the use of optochin. I therefore am inclined to the opinion that it is not the anæsthetizing but the bactericidal action of optochin which causes such a rapid disappearance of the photophobia. With what kind of bacteria, besides the pneumococci, this result is reached the future will have to tell.

As to the use of optochin we have seen that a 5 per cent. solution applied alone is felt very disagreeably and the irritation of the eye is even for a time increased, that however this irritation does not occur if it is used after the instillation of a 5 per cent. cocain solution. A 1 per cent. solution is well borne without previous cocain instillation. As to the ffect of optochin solution, we were able to determine that perfectly fresh solutions and those but a few days old gave very quick favorable results, solutions which were older than three or four weeks had lost their effect almost altogether.

A REACTION OF THE PUPIL, STRONGLY SUGGESTIVE OF ARTERIO-SCLEROSIS WITH INCREASED BLOOD PRESSURE.

Meyer Wiener and H. L. Wolfner (*Jour. Am. Med. Assn.*, July 17th, 1915) note a pupillary reaction which they think is strongly suggestive and as adding a clue in the reaching out for a diagnosis. They find that where the reaction is present the pupil is larger than the average normal pupil with a usual minimum size of 4.5 mm. to 5 mm. in width, contracts promptly to light stimulus, but immediately returns to the original size and there remains, without the light stimulus having been changed. It is not claimed that this reaction is pathognomonic of arteriosclerosis with high blood pressure. The authors maintain, however, that its association is so nearly constant as to make it a sign that is at least strongly suggestive and therefore of undoubted clinical value.

ON THE HEREDITY OF SENILE CATARACT.*

By Franz Zydek, M.D.

Although it is a praiseworthy fact that opthalmology has of late begun to show some interest in the study of heredity and the students of heredity have paid considerable attention to the affections of the eye in order to find the laws of heredity, yet there is still a great deal in this field which needs clearing up. Among the anomalies which in this direction furnish probably the largest material are the opacities of the lens which in the greatest varieties may appear in families and even through several generations. In this field much material has of late been collected by Englishmen, especially by Nettleship, but, also, by German authors. Until recently when the heredity of cataract was discussed, such a discussion usually dealt solely with the various clinical forms, as total cataract, lamellar cataract, spindle cataract or polar cataract. When, for instance, a total cataract in the later years of life was seen, the occurrence of this form as a familiar affection, was looked upon as a curiosity, without sufficient appreciation of the importance of this occurrence in clearing up the ætiological conditions. Thus the reports in literature have hitherto mostly been valuable only as casuistic communications.

Within the last few years we have become more conscious of the importance of the study of heredity and with it of the clearing up of the ætiology of the different forms of cataract. Peters,[40] in a collective paper on this subject has pointed out that the correct valuation of the conditions of heredity of cataract may give us an explanation of the manner of the development of many forms of cataract. If we leave aside the cases in which strands of mesoderm are hereditarily formed which by tearing the posterior lens capsule cause cataract formation and those forms of cataract which are inherited in association with other anomalies of the eye, for instance, microphthalmus, our interest is especially attached to the partial and total cataracts of the newly born and those in the later part of life.

As to the lamellar cataracts Peters[39] and Nettleship have shown that the old discussion whether this form develops intra- or extrauterinely, is perfectly irrelevant, if we remember that with hereditary cataracts usually a so-called "anticipation" is ob-

*Klin. Mtsbl. f. Augenhlk., LIV, p. 482.

served, that is, that the first appearance of the opacities of the lens in successive generations of one and the same family can come on every time at an earlier period of life. We need, therefore, not assume a different mode of origin for the intrauterine and extrauterine lamellar cataract, as long as they are hereditary, but we must seek the true cause of all these forms of cataract in an hereditary variation of the germ plasma which would explain not only the variation of the forms of cataract in the same family in the sense of a so-called heteromorphous heredity, but, also, exclude extraneous damaging influences. It is, therefore, as Peters has pointed out, only a quantitative difference, if in the same family lamellar cataract and total cataract are observed. If nucleus and cortex are differentiated from each other not only later in life but from the beginning, it perfectly agrees with the dogma of heredity, when Peters assumes that the variations of the germ plasma may differ within wide limits, so that all modulations between lamellar cataract and total cataract may be possible on the basis of heredity. In the discussion of these forms of cataract Peters stated that the familial appearance of lamellar cataract does not necessarily always mean a germ variation in the lens formation. For, with von Hippel,[23] we must count with the possibility that the earliest lamellar cataracts or those coming on in the first years of life may, also, be due to toxic influences, because the germ variation, also, takes in the so-called epithelial corpuscles of the thyroid and a cataract is formed on account of their diminished function, just as in diabetic cataract which appears in several members of one and the same family, we cannot speak of hereditary cataract, but only of a hereditary affection of the metabolism which leads to cataract formation.

This possibility of direct development by heredity and of an indirect one by toxic influences must be studied, also, concerning the familial appearance of cataract in the later years of life, especially the presenile and the senile forms. Besides diabetes, gout may have to be considered in the development of these cataract forms which may cause cataract by arteriosclerosis, and arteriosclerosis itself, which also may be a familial disease, and thus we can speak of a heredity of these forms of cataract later in life, only when we have been able to exclude the above mentioned indirect causes to which furthermore, as Peters states, we must reckon latent tetany which seems to play a part in presenile cataract.

As to the direct heredity of certain forms of cataract we have to reckon with the possibility that, for instance, the nuclear parts of the lens are originally formed in the germ plasma in such a labile state that their life period is shorter and thus later on the nuclear cataracts develop; or that the point underlying the heredity is a lack of vitality in the capsular epithelium which by an early death produces cortical cataract. We must, therefore, in future examine family-trees in this direction in order to get a firm basis for the question whether and in what form hereditary cataracts may appear in the later years of life.

For this necessity which results from the modern ideas about heredity the material which has hitherto been published is not sufficient. However, statements as to the heredity of these forms of cataract are not rare. It seemed, therefore, desirable to examine the existing material with the view of making it clear whether and in how far modern research in heredity could be made useful in our case.

If at first we search the text- and handbooks of ophthalmology we find a number of statements concerning the heredity of senile cataract.

Thus Juengken[28] says: "Not rarely the decrepidity in consequence of heredity is confined to the lens, while the remainder of the body is still in full action. In this way the appearance of cataract in some families at a certain period may be explained."

Chelius[12] has sometimes observed a hereditary tendency. "The cause of the development of cataract may be a faulty make up of the eye or the presence of certain dyscrasiæ."

In Arlt[3] we find the passage: "That cataract may appear as an inherited disease has been proven by the interesting observations of Beer, Lusardi and others. When cataract is observed in a family in co-ordinate or in ascending or descending line we can assume heredity only when the cataract appears in both eyes in the same form (according to my experience as a soft lenticular one) at the same period of life and not in the higher ages but in the first half of life." At the same time Arlt speaks of a case in which in one family a son at 19 and two daughters after the 20th year developed cataract.

Schauenburg[48] believes that heredity has in a few cases been proven for senile cataract.

Stellwag[53] mentions heredity among the causes for the development of cataract. "There are, however, parents free from cataract either all or most of whose children are born with cataract or are affected with it at an early period."

Becker[6] says the following: "The question of hereditary tendency to cataract might receive some enlightenment if we search the common constitutional peculiarities of the hereditarily tainted individuals. Since I know such a family in which the grandmother at 57, the father at 48 and a son at 25 developed bilateral cataract no other of the known complications being present, I cannot but think in this case of a hereditary constitutional cause." And again: "The heredity of senile cataract is of such great interest, because not the cataract but the hereditary disposition for it, that is bodily but unknown peculiarities are transmitted which favor cataract formation in the later years of life. Morgagni already mentions a family in which three sisters developed cataract while the brothers remained free. I have operated on a lady of 50 for cataract who was visited by her mother and her grandmother of over 90, one of whom had been operated on by Rosas and the other by Beer. A year ago I operated at the same time on a brother and sister for cataract."

Klein[30] mentions heredity in general among the ætiological factors of the forms of cataract and states: "That in some cases several or even all of the members of one family were affected with cataract and that, therefore, in these cases a hereditary disposition must be at work. It has as yet not been ascertained whether every case of cataract is hereditary, or whether or not what forms of cataract or certain cataracts especially are transmitted from generation to generation by heredity. Especially concerning senile cataract we can as yet not say with certainty that they are hereditary although in some families a noticeably large number of members seem to be afflicted with senile cataract, while others remain safe from it for many generations. It is, therefore, only probable and in no way certain that the senile cataract of an individual is a hereditary affection even if it belongs to a family in which cataract has often been observed, since we know that such cataracts may develop also without any known hereditary tendency."

Michel[33] says: "If heredity or a varying geographical distribution of senile cataract is emphasized, the sclerotic changes of the vascular apparatus are most apt to clear up these conditions."

Schmidt-Rimpler, also, mentions heredity among the causes of senile cataract. "In one family the mother and three sons were afflicted with cataract."

Fick[16] in speaking of the ætiology of senile cataract says: "Even the fact that senile cataract is hereditary in some families does not lift the veil from its origin."

The first larger paper on the heredity of eye affections and especially on the hereditary affections of the lens we owe to Groenouw[20] who worked up the then published material (1904). Unfortunately he does not sufficiently distinguish between the congenital and the later forms of cataract. The interesting cases will be mentioned later on.

According to Fuchs:[18] "Heredity makes its influence felt even with non-congenital cataracts, even with senile cataract; there are families in which many members (and then usually quite early) get blind from senile cataract."

Vossius[54] states that heredity can many times be proven for senile cataract. "In some families several members are affected by cataract at almost the same age."

It is astonishing that Hess[22] in his newest work in Graefe-Saemisch "On the pathology and therapy of the lenticular system" has made such an inadequate use of the previous material concerning the heredity of senile cataract.

Bach[4] states: "The tendency to presenile cataract, too, may be hereditary. As a rule only the opacity of the lens is transmitted, not the form of cataract. Collateral heredity does happen, mostly, however, it is direct." At another place he says: "Not quite seldom hereditary factors show themselves, since senile cataract occurs familially."

Finally we find in Heine[21] the following remark: "Very remarkable is, furthermore, the heredity of the different forms of cataract, about which we can hardly form as yet any theoretical ideas. Contrary to Daltonism here the direct mode of transmission with equal affection of both sexes and often through many generations is the rule."

I have purposely mentioned a number of authors who have published text- or handbooks, because we can conclude from their expressions that there are facts which are generally accepted. From this collection it is clear that senile cataract can be transmitted direct from one generation to the next generation and then usually appears as presenile cataract, while on the other hand there are, also, statements like those of Chelius, Michel, who say that it is not a direct heredity of senile cataract but the heredity of causes which dispose to cataract formation, like affections of the bloodvessels or dyscrasiæ.

We must agree that these aphoristic expressions can hardly be considered sufficient in order to assume or exclude in any case the influence of direct or indirect heredity.

Looking over the literature for communications on the heredity of senile cataract we find a series in which we can assume with more or less certainty that they speak of lamellar cataract which in former generations for some reason were operated on later in life only, not during youth, when the opacities, as is often the case, had increased. Such cases we have to exclude.

Here belong, for instance, the cases of Jonin[27] mentioned by Cahusac[10] who operated successfully in one year on mother, son and grandchild, and of Richter[42] in which the cataracts were probably also lamellar. The same holds good for the family on which Seiler[51] reports from the observations of his teacher Beer. The grandfather and father had cataract. All the children of the first wife, mostly sons, had cataract before their 12th year, while of the children with a second wife the daughters, and they alone, had cataract before their 8th year.

I select some of Cahusac's cases in which there was heredity of later forms of cataract.

Sanson[46] reports on a family in which all children became cataractous near the 24th year of life. Maunoir[32] mentions a family in which in ascendency in the father's line, the grandfather, uncle, two aunts and two female cousins had cataract, while of their four children one was born with cataract. It is not impossible that these were juvenile forms of cataract which only appeared clearly somewhat later. The further report of Maunoir, too, that he operated for cataract on three brothers at the ages of between 30 and 40 years, is too aphoristic to be of value.

Dupuytren[14] relates an observation in which a lady high in the sixties had cataract in both eyes, while her daughter became cataractous at 28, a grandchild at 17 years and two other grandchildren at even earlier dates. This seems to be an example of so-called anticipation, because we cannot assume that the grandmother had remained free from opacities if this had been a lamellar instead of a senile cataract, although we cannot altogether exclude such a possibility.

Dyer[15] saw a case of heredity of cataract in the male line through three generations, which, however, cannot be taken as proof of heredity of senile cataract. The same holds good with the cases of Bastard,[5] Streatfield[53] and Rublee.[44]

On the other hand a case of Caussade[11] may well be counted here. He observed a cataract in two sisters, one 57 the other 62 years old; in the latter it had begun 11 years previously. Mother

and grandmother, too, had been cataractous at about 50 years of age. Moreover, all the members of this family showed visual troubles in advanced age; it is, however, not stated whether these were due to opacities of the lens.

As interesting as Hirschberg's[24] observation is, it seems questionable whether it was not a hereditary lamellar cataract. In a certain family the grandfather had been operated on for cataract with success at the age of 30 and three of his 4 children became cataractous at 28 years of age, while the one year old child of the youngest daughter had in both eyes probably congenital lamellar cataracts. It is doubtful in this case whether we can speak of a hereditary presenile cataract or of a hereditary form of congenital cataract which appeared later, as in several of the previously mentioned cases. Another case of Hirschberg's[25] in which in a long-lived family all brothers and sisters had cataract around the 70th year of life, is of value for our question.

Klamroth[29] reports seven cases of cataract. In one case twin brothers became affected when 29 years old, whose father, also, had had cataract. In another case three sisters were one after the other operated on for cataract, while a cataractous brother had died before the operation. Since, however, the ages of these patients are not given, the case cannot be made use of as a proof for the heredity of senile cataract.

Appenzeller[2] gives an example of collateral heredity in a family in which of seven children of healthy parents two sisters and one brother had cataract between the 20th and 30th years of life. He further reports a case in which the mother at 54, her sons at 35 and 21 years had cataract.

Hosch[26] describes an interesting family tree. He could follow the appearance of cataract in several generations of one family. He especially emphasizes that the cataract in every succeeding generation appeared earlier than in the preceding one.

Berry[9] had similar experiences. In five generations of one family of 55 members 8 males and 12 females were affected. Thus the female sex predominated. He could further prove that the transmission took place through the female line more often than through the male line. He states the cataracts were not only congenital, but also presenile.

In the cases of Schoen[50] the simultaneous appearance of æquatorial cataract in parents and children is noticed; the latter become cataractous earlier in years from 20 to 30. According to present knowledge we have to look upon the accompanying

hypermetropia or astigmatism as simultaneous faults of development which, however, cannot, as Schoen would have it, be considered as the causes of the cataract formation.

Groenouw justly says of Fukala's[19] unsatisfactory communication that when a woman and her brother become cataractous in the 62d year of life, this with the relative frequency of senile cataract does not exclude an accidental coincidence.

Le Gendre[31] points to the cataract in the English Royal family. The Duke of Cumberland, George III, George IV, the Duke of Gloucester, the Duke of Sussex, the Princess Sophia, and the King of Hannover had cataract.

Further cases are those of Bergmeister[8] who reports senile cataract in four generations of one family, and Fouchard[17] who relates of a grandfather who at 32, the son at 26 and two children at birth had cataract. He especially mentions that it was a soft cataract, not a lamellar one.

In Schauz'[47] family three sons of a healthy mother became afflicted with quickly developing total cataracts at the ages of 26, 23 and 19. There was no other disease of the system or of the eyes present. The father, who had died when 40 years old from pulmonary disease, is said to have had poor eyesight.

Gordon Norrie[34] could follow two interesting family trees. In one family of 116 there were 30 cataracts, in the other 40 among 69 individuals and the cataract seems to have come earlier in the succeeding than the preceding generations.

Petit[41] relates of a family all the members of which got cataract at about 30 years of age, although no cause could be found for it.

Of Wood's[55] cases of some forms of cataract our interest is engaged by one family in which 11 out of 31 individuals had presenile cataract. Alvardo's[1] case which I found in Cahusac is not sufficiently plain.

Crzellitzer[13] reports on a family in which the mother had juvenile cataract at 16. Of her children the three of her second marriage had cataract at 52, 45 and 22 and a grandchild at 27.

Salus,[45] in a paper, in which he opposes Roemer's theory of the causes of senile cataract, says "the clinically undoubtedly confirmed hereditary development of senile cataract" can harmonize as well with no other theory than that it is "purely a sign of senility." It would be much easier to think of changes in ectodermal formations, as for instance, "a hereditary disposition to a regular appearance of toxins."

Rosenfeld's[43] material is not described sufficiently and the possibility exists that there are late appearing forms of congenital cataract which are observed at 20 or 30 years of age. In one case a mother, her two brothers, a daughter and a niece were affected, in the other case a woman of 32 whose father had had cataract at about the same age; the grandmother, too, had been operated on for cataract.

Aside from these smaller casuistic communications there are in literature a series of larger valuable works on this subject. Thus heredity, especially in ophthalmology, owes particularly to Nettleship a wealth of interesting material. In his first paper[35] in 1905 he brings a rich material on hereditary forms of cataract, in which he expressly states that postnatal cataracts cannot always be sharply distinguished from the congenital ones, since we can sometimes not tell whether the cataract had not been present at birth. In the chapter on "acquired or postnatal cataract" he reports on 122 families in which senile cataract was hereditary, in 375 individuals of which 145 were males and 230 were females. (In 1 generation of 32 families, 82; in 2 generations of 63 families, 152; in 3 generations of 26 families, 106; in 4 generations of 4 families, 31; in 5 generations of 1 family, 7.) In 25 families 152 individuals were affected with presenile or juvenile cataract. (In 1 generation of 3 families, 9; in 2 generations of 13 families, 35; in 3 generations of 5 families, 38; in 4 generations of 1 family, 12; in 5 generations of 2 families, 40; and in 6 generations of 1 family, 18.)

Even when we assume that among these juvenile cataracts there were some which were congenital and came into appearance only at a later time, this material is so impressive that we may draw from it conclusions as to the modus of heredity of the different forms of cataract. Concerning senile cataract Nettleship says:

(1) Senile cataract is usually hereditary in several generations without missing one.

(2) In younger generations the cataract often appears earlier (anticipation), often, however, at the same age.

(3) Senile cataracts appear and mature often at an earlier age; there are, however, great differences in the several families so that a congenital cataract may in reality be an anticipation.

These communications are enlarged and made more complete by one of 1909 which again furnishes a large material on hereditary forms of cataract. In the part on "acquired or postnatal

cataract" he reports on 77 families with 450 members of which 177 individuals (40 per cent.) had cataract. (In 1 generation of 2 families of 14 members, 9; in 2 generations of 25 families of 143 members, 59; in 3 generations of 25 families of 155 members, 58; in 4 generations of 8 families of 48 members, 21; in 5 generations of 9 families of 51 members, 17; in 6 generations of 3 families of 8 members, 4; in 7 generations of 3 families of 7 members, 3; in 2 families of 14, 6.)

He expressly states that females are oftener afflicted than males. In consequence of the prominence of anticipation the terms of "hereditary, infantile, juvenile and senile cataract" are said to be in place.

Two further casuistic communications by Nettleship may be mentioned. In the first case[36] two old married people had cataract; of their 10 children 7 became cataractous; there were, also, lens opacities among the grandchildren. Then he brought forth[37] 7 family trees of hereditary cataract; in the last four of these there were senile or juvenile cataracts in two to four generations; there was, also, anticipation.

An exhaustive study on the role played by heredity in the ætiology of cataract we owe to Cahusac.[10] He first discusses the different theories which try to explain the development of the different forms of cataract. Although we can refer a whole series of cataract forms to local and general diseases there still remains a large number which do not belong to this class. Of these certainly a large portion must be referred to hereditary influences. After some general remarks on the different modes of inheritance he reports a number of older observations from the 18th century which discuss the heredity of cataract; he, also, mentions many authors from the 19th century who speak for the heredity of senile cataract. In all of these, however, it is not senile cataract, but all forms of cataract in general so that this material collected by Cahusac cannot be made use of as far as concerns the earlier writers. The same is the case with the collection of hitherto known cases of hereditary cataract which Cahusac has made. He, also, brings a series of cases of his own. Based on his estimates he concludes that of 100 cataracts 10 are hereditary. He formulates in the main the following conclusions:

(1) Cataract may be hereditary familial disease.

(2) The influence of heredity may lead to a certain disposition of single organs to be damaged by certain influences; this damage may result in cataract. Or there is a germ anomaly.

(3) In general the cataract in the descendants appears at the same age; now and then, however, the succeeding generation is affected earlier than the preceding one.

(4) The female sex does not seem to suffer from cataract any more than the male sex.

(5) The modus of transmission is either direct or atavistic, or collateral.

Finally, we may add that Crzellitzer[13] has pointed out the great importance of an exact study of family trees. Only a biological family tree, that is, one which contains all descendants of the male as well as the female descendence, healthy and diseased, is of use if we want to gradually trace the laws of heredity. This point has later on been emphasized by Peters.

When critically reviewing the material thus collected, it is an evident fact that there is a heredity of senile cataract. It agrees with the experiences made with all other hereditary diseases of the later years of life, like for instance arteriosclerosis, that in some families the succeeding generations are attacked by cataract at even an earlier period. The presenile forms which thus appear are very numerous in our material. This anticipation of a hereditary senile cataract, as Nettleship states, can go so far that even a congenital cataract must be viewed in this sense. The material here collected proves, further, that none of these casuistic communications contain a single case of indirect transmission; for it is almost always stated that all other local or general symptoms were wanting. However, we must certainly count with this mode of development of a familial cataract appearing in several generations. Yet it remains altogether in the background in comparison with direct heredity of cataract which must be referred to a variation of the germ plasma.

If we inquire further what is the real substratum for the heredity in these directly transmitted cataracts, there are two possibilities. Either the capsular epithelium is already in the germ plasma formed so faulty that after a series of years it is no longer able to accomplish its task and that thereby the lens substance, especially the cortical layers, become opaque. The other possibility is that the lens nucleus itself has been in the same manner formed in a faulty way and that thereby its period of life is shortened. Whether these two possibilities really are present in inherited senile cataracts further experiences will have to teach. For in future in reporting such familial cataracts more importance than heretofore must be attached to the statement

of whether it was a subcapsular cortical cataract in the meaning of Hess or a so-called nuclear cataract. If it should be found that only the one or the other form appears familially we might, also, draw therefrom conclusions as to the origin of other forms of cataract; then the nuclear cataract would simply mean the death of centrally located parts of the lens, which might happen without shrinking, as we observe it so plainly in lamellar cataracts. It is, therefore, perfectly correct when Peters in his repeatedly mentioned papers demands that further material should be collected in the sense of an exact family tree study in order to get an explanation of these important questions.

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HEMERALOPIA AMONGST SOLDIERS.

An editorial in the *British Med. Jour.,* July 10, 1915, comments upon this condition. It is based upon the findings of Prof. Braunschweig (*Muench. med. Woch.,* March 2, 1915). There is an ophthalmic department attached to the fourth German army and here in a period of four weeks 22 out of 98 patients treated in hospital for diseases of the eye, suffered with night blindness. The ages ranged from 17 to 46. There was no striking relation between the health and vitality of the patients, and their symptoms. In many cases there was slight conjunctivitis or blepharitis and in two-thirds there was some error of refraction. The patients felt much better when these errors were corrected. Malingering was suspected but excluded. The treatment consisted of correcting errors of refraction, relieving the conjunctivitis and blepharitis, rest in hospital, cod-liver oil, iron and arsenic given internally, a liberal diet and uninterrupted sleep. Many of the patients traced their symptoms to the constant strain on the eyes caused by peering through the darkness in the trenches, and on the roads when driving.

MEDICAL SOCIETIES.

THE COLORADO OPHTHALMOLOGICAL CONGRESS.

Denver, 1915.

From the writer's point of view the Colorado Ophthalmological Congress held July 22d and 23d, in Denver, was an unqualified success. The attendance while gratifying should have been larger. Such meetings should receive the unreserved support of ophthalmologists the country over. While most of the large cities were represented, there were some conspicuous by their lack of representation. The situation of Denver, central as it is, blessed with unsurpassed natural advantages as to scenery and climate, should and ought to prove an ideal convention city. But above all to be reckoned was the enthusiasm, energy and true western hospitality of the Colorado Ophthalmological Society. To the Society, as a body and individually, the thanks and appreciation of the visiting members should be expressed, and in doing so the writer feels he is conveying the sentiment of each and everyone of his attending confreres. Those registered numbered about 55. The fact that the American Medical Association meeting and the American Ophthalmological Society's convention took place at dates not far removed from that of the Congress, no doubt operated to keep down a much larger attendance. The first day's proceedings consisted of the formal opening and announcements, after which the reading of the papers began. The first two papers, "Carcinoma, Apparently Primary in the Uveal Tract," by Edward R. Neeper, and "Peripheral Annular Infiltrate of the Cornea Following a Scleral Perforation," by Francis Lane, were especially interesting from the pathologist's viewpoint, and elicited some very illuminating discussion. The same can be said of Wm. E. Gamble's paper dealing with "Albinism in Man and Animals with Special Reference to the Eye." Drs. Lane and Gamble's papers were enhanced by lantern slides. "The Blind Spot," by H. S. Gradle, was so exhaustive and voluminous that Dr. Gradle confined himself to an abstract of his paper. The subject was presented in such an attractive manner that a reading of the paper in full is awaited with interest. Several of the essayists for various reasons were prevented from attending, among them H. H. Martin with a

paper on "Retino-Choroiditis Juxtapapillaris"; N. M. Black, "Protection of Eyes from Harmful Radiations in the Various Professions, Trades and Industries", and Burton H. Chance, "Herpes Zoster Illustrated by Clinical History of Five Cases".

St. Louis was represented by three papers, by F. E. Woodruff, "Complete Left Lateral Hemianopsia with Glycosuria as a Result of Slight Trauma"; Meyer Wiener, "Mydriatic Action of Dextro-Hyoscyamine", and Wm. H. Luedde, "Ocular Tuberculosis Caused by Tuberculosis of Neighboring Sinuses". Dr. Woodruff's paper evoked discussion as to the rôle of diabetes in eye diseases in general. Dr. Wiener claims for Dextro-Hyoscyamine mydriatic action without the disadvantage of cycloplegia. Unfortunate previous illness prevented Dr. Luedde from presenting his subject except in an informal way.

The complacent attitude towards so-called amblyopia ex-anopsia, the policy of laissez faire, received a merited rebuke in the excellent paper of F. Park Lewis, "Conscious Vision in the Development of Amblyopic Eye". The discussion of this paper was spirited and instructive.

While all may not agree with Robt. S. Lamb in his paper, "Is Migraine a Forerunner of Glaucoma?" yet his contention must receive consideration. As he expressed it, he is himself convinced of the relationship between these two conditions and brought the matter up in order that others may give the subject critical thought and attention. There are two subjects the discussion of which is always sure to arouse attention, viz.: glaucoma and cataract. The papers by H. W. Woodruff, "Trephining Versus Iridectomy in Glaucoma"; "Mule Shoe Drain for Chronic Glaucoma", by A. E. Prince; "Arrest of Cataract at an Early Stage", by E. L. Jones; "Management of Eyelids During Cataract Operation", by D. T. Vail, and "The Routine Treatment of Senile Cataract by Expression with a Sliding Conjunctival Flap", by Harold Gifford, proved no exception to this rule. Trephining while having its advocates would probably, had a vote been taken, have lost to iridectomy. Dr. Woodruff inclined towards iridectomy as the operation of choice, especially so in acute glaucoma. Dr. Prince's mule shoe drain has for its purpose the maintenance of the patency of the trephine opening.

Dr. Jones' paper follows much the same lines as the writings of Col. Smith who recommends cyanide injections in early cataract. Dr. Vail, with lantern slides, illustrated his method of controling the lids by the use of lid retractors of his invention.

Those who advocate an intra-capsular operation must of necessity end the section in clear cornea. Many who do the ordinary extraction prefer to come out in the cornea feeling that the disadvantages of the procedure are more than counterbalanced by the disadvantages of a flap. However, most ophthalmologists will agree with Dr. Gifford that the security afforded by a flap is of paramount importance. Dr. Gifford pointed out the still greater advantage which accrues from a sliding flap, which in addition to warding off the chances of infection acts as a consistent barrier to vitreus and iris prolapse.

H. Moulton's article on the "Use of Lead Styles in the Treatment of Stenosis of Lacrimal Duct," was a reiteration of the procedure which he has long advocated in the treatment of this most chronic and troublesome condition. He took up seriatim the objections to the method and pointed out how they might easily be obviated.

Dr. D. F. Harbridge reported a case of "Traumatic Rupture of Eyeball, Complete Aniridia; Preservation of Lens with Practically Normal Vision." His paper included a review of the literature and the cases previously reported.

"Concerning the Surgical Treatment of Retinal Detachment," by E. S. Thomson, evoked great interest, as would be expected from any method likely to improve results in this ophthalmological bete noir. While the end results in his cases leave much to be desired, yet there is a gleam of hope that the method, if consistently carried out in selected cases, will offer more in the cure of this hitherto hopeless condition than any procedure has in the past. It has logic and sound surgery for its basis.

Fate decreed that the 13th paper on the program deals with the only subject not strictly a scientific one. It seemed somewhat out of the usual to have in a scientific meeting a paper dealing with the business side of medicine. Such was the article by Dr. Linn Emerson, on "Business and Office Methods in Special Practice". While it is often true that scientific men are poor business men it does not follow that good business men are poor scientific men. There can be no objection to any method which expedites the handling of patients or lessens the burden of work on the physician, especially so if in the end it redounds to the benefit of the patients. And if it adds to the prestige and pecuniary gain of the doctor, so much the better for the system. With his usual becoming modesty Dr. Jackson had himself placed last on the program. His subject, "Juvenile Recurring Hæmor-

rhage, Massive Exudation and Tuberculosis of the Retina", because of the short time at his disposal, was abbreviated and but the main points touched upon.

Dr. Jackson's paper concluded the scientific program. After adjournment the visiting members were taken for a ride through the mountain parks in autos, ending at Morrison, where a dinner brought the meeting to a close.

W. F. HARDY.

TRANSACTIONS OF THE PHILADELPHIA POLYCLINIC OPHTHALMIC SOCIETY.

March 11, 1915.

SYMPOSIUM ON GLAUCOMA.

"The Pathogenesis and Ætiology of Glaucoma."—Dr. Samuel D. Risley (by invitation).

Dr. Risley said that he knew of no chapter in ophthalmology which had a wider literature than this and in which so little general advance had been made until within a few years, and so far, he believed, much of the delay in a definite judgment and opinion with reference to this disease was due to the name, that is to say, the term "glaucoma". It should be studied from the standpoint of the symptom complex and what it stands for, increased tension of the eyeball. For a number of years he has been very much interested in the study of the uveal tract of the eye, and as long ago as the time of the late Dr. Shakespeare when the talks they had on the subject of the investigations to be made along the line of the choroidal tract led to the latter inventing the microscope.

Dr. Risley said that to him increased tension of the eyeball still constituted one of the most perplexing and discouraging chapters in ophthalmology. He said that he would like to dismiss the term glaucoma and study the significance of increased tension of the eye ball. After quoting from his article, "An inquiry regarding increased tension of the eyeball," which he read before the American Academy of Ophthalmology and Oto-Laryngology in 1913, Dr. Risley said that the great question was the ætiological factor.

If we pass rapidly before our minds the considerable group of ocular conditions which either culminate in increased tension or threaten to do so, we will be impressed by certain significant facts. For example, if we consider the atypical forms of glaucoma, (a) the buphthalmic eye of infancy and young childhood; (b) the cases of serous iritis; (c) chronic recurrent iritis of the plastic type, in each we find increased tension, seriously impaired acuity of vision, and when possible to determine it a contracted field, and in the laboratory cupped optic nerve, frequently the signs of an optic neuritis, choriocyclitis, iritis, atrophied ciliary muscle, dilated ciliary vessels, a degenerated vitreous body and an opaque lens.

Another atypical group is constituted by perforated cornea from corneal ulcer, or by perforating and infected wounds, leading to a partial corneal staphyloma, empty anterior chamber, iridocyclitis, increased tension and loss of vision. In the laboratory cupped optic nerve, inflamed and swollen, turged or atrophic ciliary muscle, dilated ciliary vessels, degenerate vitreus and opalescent or opaque lens.

Approaching the usually regarded typical forms, that is to say, the so-called primary glaucoma, our minds at once revert to their usual grouping: (a) Glaucoma simplex or the so-called non-inflammatory type, regarding the nature of which there has been so much discussion. (b) The subacute inflammatory type, beginning with transient impairment of vision, recurring attacks, increasing in severity and with briefer intervals between the exacerbations, the inflammatory features and pain meanwhile growing more pronounced, the anterior chamber shallow, the cornea steamy, the iris atrophic; the impairment of vision, the contraction of the field and increased tension, at first transient but becoming permanent. The anterior perforating vessels (long anterior ciliary) become permanently engorged; and from the first the transient attacks are associated with impaired range of accommodation. If neglected total blindness ensues and the disease passes into a stage of absolute glaucoma. In the laboratory is found a deeply cupped disc, atrophic optic nerve, atrophic ciliary muscle with evidence of preceding inflammation, dilated ciliary vessels, degenerated vitreous body, opaque lens; invariably both eyes in near sequence are involved. (c) The acute inflammatory type more violent from the onset, the final rseult as set forth in the former group rapidly consummated and the same laboratory findings in more exaggerated form.

(d) the so-called hæmorrhagic glaucoma, constituting one of the most striking and hopeless forms of the disease with which the ophthalmic surgeon meets.

He has in this summary eight well defined groups, characterized by increased tension of the ball and its disastrous sequelæ and by certain pathological findings in the laboratory more or less common to them all. What relation if any do they sustain to each other? Is there some ætiologic factor, more or less common to them all, to which the increased tension is secondary?

In discussing these inquiries there are certain general considerations that cannot be evaded. If we take for example the buphthalmic child with its destructive uveal disease, beginning before birth or during infancy or early childhood, we find it is obviously dependent on or constitutes a part of some general, usually hereditary disease. He has published a striking example of the latter under the caption of prenatal iritis in a syphilitic child, in which the buphthalmos occurred and the child passed into imbecility, the victim of syphilitic endarteritis.

In conclusion Dr. Risley said that treatment of glaucoma or increased tension of the eyeball must be addressed to the ætiological factor, which, if done, alone holds out hopes of recovery.

Dr. Posey spoke on the use of miotics in the treatment of chronic glaucoma. He cautioned, however, that this form of treatment was applicable only to cases in which there were no inflammatory symptoms, as the presence of these later demand some form of immediate operation. Miotics to be effective must be employed persistently and continuously four times a day. Their effects pass off at the end of three hours, and if the maximum amount of contraction of the pupil is to be maintained, the reinstillation must be faithfully carried out. In beginning cases the dose of the miotic should be weak, to avoid creating spasm of the iris or ciliary muscle, the strength being increased from time to time as the drug loses its effect upon the pupil. Dr. Posey said he employed the nitrate of pilocarpine at 8 a.m., 1 and 6 p.m., but ordered the salicylate of eserine for the last instillation at night, on account of the more lasting effect of the latter drug. To obviate the irritation set up sometimes by the miotics, he spoke of the necessity of flushing out the conjunctival cul-de-sac some ten or fifteen minutes before the miotics were employed, with boracic acid solution. All miotic solutions should be compiled fresh once a month and should be perfectly sterile. Care should be taken to keep the dropper sterile. Attention

should be given to the general health, tobacco prohibited and strychnine and alteratives prescribed for their effect on the optic nerve. He quoted figures deduced by him which showed far greater success from the treatment of miotics than from operation. He thought the treatment applicable to all stages of chronic glaucoma. He advised operation, however, on all hospital cases, on account of the difficulty of keeping this class of cases under continuous observation.

DISCUSSION.

In closing Dr. Posey said that while the persistent, continuous use of miotics is a nuisance, nevertheless they never did any harm, whereas even the most skilfully performed iridectomies frequently interfered with vision by the creation of opacities in the lenses in consequence of hæmorrhage from the iris at the time of the iridectomy.

THE OXFORD OPHTHALMOLOGICAL CONGRESS, 1915.

It speaks volumes for the energy of this Congress that the annual meeting was held on July 15th and 16th last at Keble College, Oxford, under the mastership of Mr. Sydney Stephenson, much as though Europe were not plunged into a calamitous war. *Mais la science n'a pas des frontières.*

The feature this year was the discussion on "Industrial diseases and accidents," opened by Dr. Frank Shufflebotham, of Newcastle-under-Lyme. Among the numerous communications on miners' nystagmus, industrial cataract, plumbism as it affects the eye, siderosis, special mention should be made of one contributed by Dr. Edgar L. Collis, of the Home Office, on "Eye injuries caused by occupation; their prevention and first-aid treatment." The discussion on these various subjects was maintained with great vivacity by the members. The presence of Dr. T. M. Legge, of the Home Office, added much interest to the debate, and elicited many interesting facts from his unrivalled experience of industrial conditions.

Sir St. Clair Thomson, of London, gave an interesting lantern address on the surgical anatomy of the nose, and the accessory sinuses, in relation to the eye and orbit. Sir Mackenzie Davidson, also of London, demonstrated the use of the telephone detector for foreign metallic bodies in and about the eye, more

especially in the orbit. Mr. W. H. H. Jessop had an opportune paper dealing with the question of papillœdema following injuries of the vault of the skull in the present war. Mr. Stephen Mayou delivered an address on "Mooren's ulcer of the cornea," and showed excellent pathological specimens of that rare disease upon the screen. Lieut.-Col. R. H. Elliot gave an address upon the operation of couching, as practised in India, and demonstrated a remarkable series of eyes lost as the result of that ancient procedure. The discussion went to show that in a small class of cases there was still room for "couching" performed under modern aseptic conditions. Mr. T. Harrison Butler's paper on "The tragedy of sclerostomy", which gave rise to an animated discussion, is printed in the August issue of *The Ophthalmoscope*.

In the scientific museum there were exhibits by Mr. Sydney Stephenson and Lieut.-Col. Elliot, F. W. Edridge-Green, J. Burdon-Cooper, Percival J. Hay, R. J. Coulter, W. H. H. Jessop, Philip H. Adams, N. C. Ridley, John Gray Clegg, C. J. Russ Wood, S. E. Whitnall, J. H. Sutcliffe, Robert Fagin, of Memphis (*in absentia*).

At the Eye Hospital cases were shown and operations demonstrated by Mr. Philip H. Adams, Dr. William Robinson, Mr. N. C. Ridley, Mr. E. H. E. Stack, and Mr. Bernard Cridland, the honorary secretary of the Congress.

A word should be added about the social side of the Congress, which, as usual, was well maintained. Mr. Robert W. Doyne, the past Master of the Congress and its founder, presided at the dinner of the Congress, which was held on the 15th of July in the Hall of Keble College. On the 16th there was to be a garden party in the gardens of Exeter College, but since the elements were unkind, the members of the Congress were received by the Rector of Exeter and Mrs. Farnell in the College Hall. The Rector was good enough to explain the various points of interest, not only of the Hall, but of the College Chapel as well. That evening there was a smoking concert in the junior common room at Keble College.

For the first time in the history of the Congress, to the regret of everybody, not a single American citizen was present. Foreigners, too, were conspicuous by their absence. What the meeting lacked in numbers, was, however, more than compensated by the enthusiasm of the members. The informality of all proceedings was, if possible, more marked than ever.—*The Ophthalmoscope*.

ABSTRACTS FROM MEDICAL LITERATURE.

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THE EFFECT OF MOVING PICTURES ON THE EYES.

In a symposium dealing with this subject (*The Medical Times*, July, 1915), contributed by J. Norman Risley, J. Herbert Claiborne, J. Morrison Ray, Seth Scott Bishop and Dunbar Roy, the question of the harmful effects of moving pictures on the eyes is discussed. The "flickering" and bright flashes of light are considered detrimental due to a retinal congestion induced. Eyes with refractive errors, as one would suppose, are the greater sufferers from ocular discomfort provided that the error is not so great that no effort of accommodation is able to produce a clear retinal image. These latter cases in every day life we know often have no ocular discomfort. Muscular imbalance is also conducive to aggravated asthenopia resulting from moving pictures. Claiborne does not think that moving pictures *per se* are harmful to the eyes and gives the film productions his hearty approval.

It seems that persons adversely affected by moving pictures would be caused to suffer ocular discomfort by any other prolonged and steady fixation. Again, the headache consequent upon attendance at a picture show may be due to poor ventilation. No one seems to have any clinical data on the subject. As Roy states, the question of the effect of moving pictures on the eyes is very much like the question as to what effect does the eating of cabbage have upon the digestion. The answer is that it affects different individuals in different ways. When a person realizes that his or her eyes are fatigued, burn, become red, water easily, and possibly have a dull frontal headache after watching a picture performance, it is indicative that to these people such amusements are injurious to the eyes.

This train of symptoms occurring under any other conditions would call for an ophthalmological examination. It would seem good policy then for individuals so affected by moving pictures to have their eyes and ocular muscle apparatus looked after by a competent oculist.